ON THE EPIDEMIC ACUTE AND SUBACUTE NON-SUPPURATIVE INFLAMMATIONS OF THE NERVOUS SYSTEM PREVALENT IN THE UNITED STATES IN 1918-1919: ENCEPHALITIS; ENCEPHALOMYELITIS; POLYNEURITIS; AND MENINGO-ENCEPHALO-MYELO-NEURITIS.[1]

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The Clinical Phenomena of the Disease.

This epidemic disease gives rise to clinical phenomena that are referable chiefly to disturbances of the functions of the nervous system. Since any area in the nervous system may be involved, with either loss of, increase of, or perversion of function, and since the single cases vary greatly as to the number and distribution of areas diseased as well as to the intensity of the process in particular areas, we may, in epidemic encephalitis, meet with the greatest variety of neurological syndromes, so that it is not surprising that some clinicians, overlooking an underlying unity, have thought that several of the syndromes correspond to as many different and independent diseases. Just why the intensity of the process and the distribution of the injuries to the nervous tissues are so variable we do not yet know, but there is good reason to believe that in this epidemic we are dealing with a single infectious agent as a cause, and that this agent attacks the central and peripheral cerebrospinal nervous system and its coverings in a widespread, though, at the same time, in a patchy or disseminated way. Under such conditions a great variety of combinations of general and focal symptoms must be expected in a series of cases. An understanding of the underlying pathological-anatomical process in epidemic encephalitis makes clinical diversity no surprise; what arouses wonder is rather the frequency with which certain associations of symptoms, such as lethargy, with fever and opthalmoplegia, are met with in the epidemic. (See Table I.)

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[1] This paper was presented (in abstract) before the Association of American Physicians at its meeting at Atlantic City, in June, 1919, and the abstract will be published in the Transactions of the Association.

TABLE I.—CLINICAL PHENOMENA OBSERVED

Case and date examined, 1919.	N. II. Opticus.	N. III (oculomotorius).				N. IV. Trochlearis	N. V. Trigeminus.	N. VI. Abducens.	N. VII. Facialis.
		Ptosis.	Ext. bulbar muscles.	Iris.	Accommodation.				
I. C. M. C. Feb. 28	0	Partial bilateral	All weak	Sluggish; anisocoria	....	....	Partial paralysis and sensitiveness of rt. masseter; deflection of jaw to rt.	Paresis of right M. rect. lat.	Bilateral facial paralysis
II. K. S. C. Feb. 28	0	0	0	Sluggish pupils; anisocoria	Difficulty reading	0	0	0	Bilateral paresis and mask
III. N. M. L. Mar. 2	0	0	0	Sluggish; pupils sl. irregular	....	0	0	0	0
IV. K. L. Mar. 13	0	0	0	Miosis and slight irregularity	....	0	0	0	0
V. F. I. Mar. 17		Partial bilateral	Weakness, especially of left M. rect. med.; diplopia	0	Inability to read	0	Sensitiveness of masseters (Thomas's sign).	0	Tremor of upper lips; loss of expressive movements with bilateral paresis of face muscles
VI. D. O. H. Mar. 24	0	0	0	Slight anisocoria and sluggish rt. pupil	....	0	0	0	0
VII. F. X. Mar 22	0	Slight right	Diplopia; weakness of all mm. especially superior rect.	Sluggish pupils	Inability to read for week before admission	Bilateral paresis	Bilateral dysmasesia; tender masseters on ninth day.	Bilateral paresis	Bilateral paresis of lower right facial
VIII. F. M. T. Apr. 14	0	0	Diplopia	Sluggish pupils	Temporary paresis	0	0	Paresis on left side	0

IN CASES OF EPIDEMIC ENCEPHALITIS.

N. VIII (acusticus). Cochlearis. Vestibularis.	N. IX. Glosso-pharyngeus.	N. X. Vagus.	N. XI. Accessorius.	N. XII. Hypoglossus.	Other phenomena.
0	0	Pulse-rate 108	0	0	Undernourished; gastric hyperacidity; 30 small mononuclear cells in c. s. f. with ++++ globulin and negative Wassermann.
Tinnitus	0	Pulse-rate 104	0	0	Pain in head and neck; nervousness, emotionalism and weakness; aching pains in legs; insomnia; forgetfulness; irritability; difficulty in walking; mental depression; well-marked Parkinsonian mask; marked drowsiness at onset; rather marked, coarse tremor of head and trunk; finer tremor of fingers; systolic pressure 190, diastolic 120; oral sepsis; slight hyperthyroidism; chronic alcoholism, tabagism and strychninism.
0	0	Pulse-rate 90	0	0	Brudzinski +; Kernig +; Babinski equivocal on left; delirium; history of potatorium; in c. s. f. 133 small mononuclear cells; strongly positive globulin and negative Wassermann; "luetic type" of curve with gold sol. test; alb. and casts in urine with phthalein output of 70; 35 pounds underweight; improvement after lumbar puncture; slight fever.
0	0	Pulse-rate 104	0	0	Reflexes normal; no objective disturbance of sensation, but complaint of persistent numbness and tingling in left arm, left loin and left leg, which came on two days after an attack of influenza, and has progressively increased in intensity; ten days after onset, transitory glycosuria; c. s. f. showed 30 small mononuclear cells, positive globulin and negative Wassermann; moderate obesity and slight arterial hypertension; marked oral sepsis.
0	0	Pulse-rate 112	0	Marked tremor of tongue	Marked Parkinson mask; pronounced apathy and depression; delirium at onset after influenza; c. s. f. showed 7 small mononuclears, positive globulin; negative Wassermann; atypical paretic curve with gold solution test; achylia gastrica; myoma uteri; undernutrition 25 pounds; temperature 99 to 100°.
0	0	0	0	0	Onset with backache, hiccough and girdle pains; later severe root pains in lower extremities and later in chest; slight fever; marked drowsiness for 4 or 5 days; unsteady gait; insomnia; c. s. f. showed increased pressure, 48 small mononuclear cells; globulin +++; Wassermann negative; improvement after lumbar puncture; fluid a week later showed 14 small mononuclear cells and ++++ globulin; positive Oppenheim on right.
0	Slight dysphagia	Pulse-rate 100	0	0	Slight fever; drowsiness; unilateral sweating of face; lacrymation; headache; mask-like face; c. s. f. bloody but negative Wassermann; slight nystagmus.
0	0	Pulse-rate 84	0	0	Stupor at onset; long standing psychasthenic state; marked oral sepsis; anemia; slight fever; undernutrition.

ONSET. This may be sudden or gradual, and with or without prodromata (asthenia, lassitude, headache, vomiting). Some of the patients regard the attack as influenzal; others give a history of having had influenza a few days or a few weeks before; still others assert that they have not had influenza at all. The general encephalic symptoms may, in some cases, be the more marked at onset; in others, focal symptoms, referable to brain or spinal cord, or signs of meningeal irritation, announce the presence of a nervous disease.

GENERAL SYMPTOMS. The most striking general symptom, when it is present, is a disturbance of the general consciousness that manifests itself in a pathological drowsiness, a symptom that led von Economo to call the disease "encephalitis lethargica," and the lay press to dub it "sleeping sickness." All grades of this disturbance (apathy, dulness, somnolence, sopor, stupor, coma) may be met with, either in a single case or in a series, and either at or near the onset or later in its course. Some patients, however, do not manifest the symptom at all. In one patient of my series, who was drowsy for about a week, the tendency was attributed at first, erroneously, to the effects of codein administered for the relief of pain. Milder involvements of the general consciousness (somnolence and sopor), from which the patients can be roused and made to answer questions, seem to have been more common in the epidemic than the severer disturbances (stupor and coma).

Other disturbances of the general consciousness, including mental depression, anxiety and delirium, also frequently occur. A patient may be drowsy in the daytime and complain of insomnia and restlessness at night.

Headache, vertigo, tachycardia and vomiting are common general symptoms. Outspoken choked disk was not present in any of the cases of my series, and, judging from the reports, is not met with, though a slight optic neuritis may occur. Fever may or may not be present at the onset, but is usually evident at some time during the course of the disease. It was slight in most of the cases we have seen, though we were impressed with the persistence of an elevation of temperature of 1° or 2°, extending over a period of from one to three weeks in several of the cases. In some cases there is marked pyrexia, 102° to 105°, lasting for from one to a few days.

FOCAL SYMPTOMS. These are predominantly motor rather than sensory.

Commonest by far are bilateral nuclear and radicular paralyses of the eye muscles (of well-known type), with ptosis and ophthalmoplegia externa et interna (polioencephalitis superior acuta involving N. III, N. IV and N. VI); but pontile and bulbar nuclear and radicular paralyses, with facial paralysis, dysmasesia, dysphagia, dysarthria, etc. (polioencephalitis inferior acuta involving N. V (motor), N. VII, N. IX, etc.), are also common, as are symptoms pointing to paralysis of part of the extrapyramidal motor system

(Parkinsonian mask; loss of the normal facial innervations accompanying the emotions). The lesions that are most frequent as causes of motor focal symptoms in epidemic encephalitis must therefore be located in (1) the midbrain about the aqueductus cerebri, (2) the pons and upper medulla oblongata and (3) the basal ganglia (globus pallidus?).

Less common motor disturbances in epidemic encephalitis, though they may occasionally be encountered, are monoplegias, hemiplegias, diplegias, aphasias, contractures, choreatic and athetotic disturbances of motility and general or circumscribed convulsive seizures.

Focal phenomena of a sensory nature are much less frequent than those of a motor nature above referred to. Nevertheless, sensory symptoms due to focal lesions of the sensory areas (cortex) and paths (corona radiata; lemnisci) are occasionally met with (hemi- or mono-anesthesias; paresthesias; hyperesthesias; algias; more rarely, hemianopsia). In one of our cases, in which there was a meningomyelo-encephalitis, severe root-pains in the trunk and lower extremities were prominent.

Acute ataxias of different types (according to the localization of the lesions) may be exhibited. The commonest form is the cerebellar type, due doubtless to lesions of the cerebellum or its peduncles or of the red nucleus mechanisms.

MENINGEAL SYMPTOMS. Only in relatively few cases are the clinical signs of an outspoken meningeal irritation present. In one case of our series there was pain in the neck and back, some rigidity of the neck, a positive Brudzinski sign and a positive Kernig sign. von Economo, S. A. K. Wilson and others have reported similar cases. That there is a slight patchy involvement of the leptomeninges in the inflammatory process in many cases has been shown, however, by postmortem examinations and by the increased cell count and the positive globulin reaction in the cerebrospinal fluid.

CEREBROSPINAL FLUID. This may or may not be under increased pressure. The fluid looks perfectly clear, and no film, as a rule, forms on standing. In some undoubted cases of the disease the cell count has been normal and the globulin test negative. The apparent normality of the fluid on naked-eye examination apparently misled many of the European observers, who made the earlier reports on the disease, to declare that no changes are present in the cerebrospinal fluid. Abnormal fluids have been very constantly encountered in our own cases, though they would have been overlooked because of the apparently normal macroscopic appearances had the examiner failed to make a cell-count and a globulin test. *In our experience, a cell-count in the cerebrospinal fluid of from* 10 *to* 100 *small mononuclears along with a positive globulin reaction with negative Wassermann and negative bacteriological smears and cultures* *is, at the time of an epidemic of encephalitis, strong corroborative evidence of the existence of the disease in a patient in whom the process is for any other reason suspected to exist.* (See Table II.)

TABLE II.—CEREBROSPINAL FLUIDS IN EPIDEMIC ENCEPHALITIS.

Case.	Day of disease.	Appearance.	Pressure.	Globulin.	Number and variety of cells.	Wassermann reaction.
I. C. M. C.	19th	Clear, colorless	.....	++++	30	0
II. K. S. C.	106th	Clear, colorless	Increased	++++	11	0
III. N. M. L.	21st	Clear (fairly)	Increased	++++	97	0
	23d	?	?	?	Increased; 98% lymph., 2% leuko.	?
	26th	?	?	++++++	133	0
	75th	Clear	Increased	+	33	0
IV. K. L.	66th	?	Normal	+	30; lymphocytes predom.	0
V. F. I.	51st	25 c.c. cl. fluid	Not increased	+	2; lymphocytes	0
	53d	?	?	Weak; pos.	?	0
	66th	?	?	++	7	0
VI. D. O. H.	29th	Clear, colorless	Much increased	+++	48; sm. monos.	0
	36th	Clear, colorless	Much increased	++++	14; sm. monos.	0
	41st	Clear, colorless	Increased, but not so much as formerly	++++	14	0
VII. F. X.	7th	Bloody	?	?	Bloody.	0
	13th	Bloody	Not increased	?	?	?

Negative findings in the cerebrospinal fluid do not, however, rule out the disease.

Still another point regarding the cerebrospinal fluid is worthy of comment. In more instances than accidental injury to a small bloodvessel will apparently account for, a bloody fluid is obtained on lumbar puncture in patients suspected to be suffering from the disease. This point has been noted also by other observers, and it seems likely that the well-known hemorrhagic tendency in the disease is thus sometimes manifested.

TABLE III.—BLOOD IN EPIDEMIC ENCEPHALITIS.

Case.	Day of disease.	Red blood cells.	Hemoglobin, per cent.	White blood cells.	Polymorphonuclear neutrophiles, per cent.	Polymorphonuclear eosinophiles, per cent.	Polymorphonuclear basophiles, per cent.	Small mononuclears, per cent.	Large mononuclears and transitionals, per cent.	Wassermann.
I. C. M. C.	21st	5,212,000	90	6,200	62.0	2.0	0.4	23.2	12.4	0
II. K. S. C.	105th	5,536,000	95	12,800	87.2	0	0	9.6	3.2	0
III. N. M. L.	19th	4,844,800	90	22,850	90.0	1.0	1.0	7.0	1.0	0
IV. K. L.	60th	5,472,000	90	10,000	76.0	0.4	0.0	17.2	6.4	0
V. F. I.	42d	5,856,000	85	10,000	82.8	0.0	0.0	12.8	4.4	0
	57th	?	90	7,700	?	?	?	?	?	?
VI. D. O. H.	30th	4,800,000	85-90	9,000	?	?	?	?	?	0
VII. F. X.	7th	6,328,000	77	9,120	75.5	1.0	0.0	16.0	7.5	0
	9th	6,512,000	75	9,820	76.0	1.0	1.0	13.0	9.0	
	19th	5,280,000	78	7,800	75.0	0.7	0.0	18.7	5.7	
VIII. F. M. T.	30th	5,408,000	97	17,600	70.4	0.4	0.4	22.8	6.0	0
	62d	3,600,000	70	3,058	72.4	1.2	0.0	18.0	8.4	

THE BLOOD. A slight leukocytosis is usually present. The white cell count in our cases varied between 3058 and 17,600. per c.mm., except in one instance in which the cell count was 22,850. The differential count of the white cells usually, though not always, reveals a slight relative and absolute increase in the polymorphonuclear neutrophile elements (70 to 90 per cent. of the total white count). (See Table III.)

THE URINE. A trace of albumin and a few casts are sometimes demonstrable, doubtless due to a slight toxic degenerative nephropathy such as is met with in most acute infectious processes. Renal function, as far as the phthalein test has been applied, appears to be unimpaired. When severer renal disease, as in chronic alcoholism, accidentally accompanies encephalitis with lethargy, the differential diagnosis may be difficult as in one case of our series in which the delirium and coma were thought, at first, to be due to uremic intoxication.

DURATION OF THE DISEASE. Whether the disease terminate in death or in recovery, the course may be either brief or prolonged. In severe fulminant cases death may occur in a few hours or days. In many instances, both mild and severe, recovery has been rapid, the symptoms lasting from a few days to a month. In the majority of cases, however, the disease is a protracted one, extending over many weeks, and in some instances over several months.

PROGNOSIS. As regards life this is much better than might have been expected, even in cases that at onset present the gravest symptoms. The mortality has been variable in different countries, and seems to have been greatest in Austria and in France. Six out of 11 of the cases reported from Austria by von Economo died; 7 of Netter's 15 cases reported from France were fatal; only 2 of S. A. K. Wilson's 13 cases reported from London were fatal; in our own series it happens that there have been no deaths at all, due to accidental encounter of a mild group of infections. Though just what the mortality rate has been in the United States it is as yet difficult to estimate. Bassoe, Pothier, and Tucker have all reported fatal cases, and the newspapers have given evidence of many deaths all over the country from "sleeping sickness."

As regards residues and sequelæ in the patients who recover, it is, as yet, of course, too early to make any definite statement. We have been impressed, on the one hand, with the very remarkable improvement observable in some instances within a few weeks (disappearance of paralyses; recovery of facial expression and vanishing of the Parkinsonian mask; replacement of depression by normal mood), and, on the other hand, with the persistence in some cases over a long period of the eye-muscle disturbances, the dizziness, the depression and the asthenia.

We must be on the lookout in the coming period, we believe, for sequelæ, especially, as it would seem likely that large numbers of the milder infections must have gone entirely unrecognized. We have the fear, let us hope that it is not well-founded, that, as a result of the epidemic, we may see an increased incidence, later on, in the conditions hitherto known as "multiple sclerosis," unexplained "mental deterioration," and "idiopathic epilepsy."

Nature of the Disease.

Observers had but little difficulty in determining early in this epidemic that they had to deal with an infectious, or toxi-infectious, process involving predominantly the cerebrospinal nervous system. The clinical phenomena of an epidemic disease (of acute onset with fever) that manifested itself by a disturbance of the general consciousness and by paralyses, especially in the domain of the upper, and sometimes of the lower, cerebral nerves left no doubt on this point; but had there been any doubt it would soon have been dissipated by the necropsy findings, which revealed lesions of an inflammatory, and sometimes of a hemorrhagic, character disseminated throughout the nervous system and its coverings (encephalitis; encephalomyelitis; meningo-encephalomyeloneuritis). The resemblance of the clinical features in single cases to various well-known and less well-known syndromes (Wernicke's "polioencephalitis superior acuta;" Strümpell's "primary acute encephalitis;" Wickman's "encephalitic form of the Heine-Medin disease;" Leichtenstern's "post-influenzal encephalitis;" "acute infectious polyneuritis;" botulism with ophthalmoplegia; the mysterious epidemic disease known as "nona" in 1890; and the epidemic "sleeping sickness" (*Schlafkrankheit*) described after a grippe epidemic in Tübingen by Camerarius as far back as 1718) was quickly detected by one or another of those who had opportunity to study the outbreak. An epidemic of an acute infectious (and inflammatory) disseminated meningo-encephalomyeloneuropathy closely resembling, if not identical with, certain diseases earlier described had, then, presented itself in many different countries of the world for study and for consideration.

Concerning the etiology of the disease, very different opinions have been advanced. Some investigators, especially those who studied in the first two or three areas of what is now seen to be a pandemic distribution of the disease evidently thought that they were dealing with an entirely new malady. Thus, von Economo, who, in Austria, described it as "encephalitis lethargica," Bradford, Bashford and Wilson, who studied a special group of cases in the British Expeditionary Force and designated it "acute infectious polyneuritis," and Netter, who in Paris described it as "encéphalite léthargique épidémique," all seem inclined to regard it as a new disease, though they admit its resemblance to recognized diseases. It was soon seen, however, on analysis of the clinical phenomena and the pathological-histological findings, that under some one or other form of encephalitis, or of encephalomyelitis, parallel phenomena have already been described in medical literature. Thus most of the phenomena, both clinical and histological, will be found paralleled in the larger collective reviews of acute non-suppurative encephalitis, such as those by Chartier (1907), by Oppenheim and Cassirer (1907) and by Vogt (1912). Most of the cerebral-nerve paralyses will be found duplicated in the cases of encephalitis tabulated by Wilbrand and Saenger (1900). Most, if not all, of the pathological-histological findings appear to be in accord with those described by Friedmann (1890–1898), Bücklers (1892), Flatau (1900), Spielmeyer (1902) and Raymond and Cestan (1904). One of the English writers (S. A. K. Wilson) concludes that "what appears to be new is the occurrence in epidemic form of cases of encephalitis not to all appearance due to the action of a known infective or toxi-infective agent, but presenting clinical symptoms any or all of which have been noted in various kinds of encephalitis with which the neurologist has long been conversant." But is even the occurrence in epidemic form new? Have we not records of one epidemic by Camerarius in 1718, of another by Lepecq de la Clôture in 1763, of at least three mentioned by Ozanam (in his history of epidemic diseases) as occurring in 1745, in 1800 and in 1802, of an epidemic of encephalitis described by Leichtenstern after the great influenza epidemic of 1889–1890, as well as of epidemics of "nona" that occurred at about the same time (reports collected and reviewed by Longuet in 1892)? In England the first cases observed in the present epidemic of encephalitis were thought to be instances of meat-poisoning (botulism). The problem of etiology as at present discussed for the recent epidemic resolves itself into three questions: (1) Is the disease due to the virus that causes influenza? (2) Is the disease due to the virus that causes the Heine-Medin disease or to a modification of that virus? and (3) Is the disease due to a virus *sui generis?*

The possibility that the virus that causes influenza may also be the cause of the encephalitis recently pandemic is well worthy of serious consideration; for we have good reason to believe that encephalitis, like meningitis, like pleuritis, like nephritis and like other inflammations, may at different times be due to entirely different causative agents. Thus it is well-known that non-suppurative encephalitis may occur as a complication of scarlet fever, of measles, of chicken-pox, of whooping-cough, of pneumonia, of anthrax, of malaria, of poliomyelitis and of other infectious diseases, and it is probable that these complicating inflammations of the brain are due to local infection of the nervous system with the specific virus that causes the primary general disease rather than that they are all instances of mixed infections of a unitary virus causing encephalitis and of the specific viruses of the several infectious diseases mentioned. Now if this view be a correct one it is surely not a wholly improbable supposition to suggest that an epidemic, or a pandemic, of encephalitis occurring in association with or soon after an epidemic, or a pandemic, of influenza may be due to an encephalic localization of the influenzal virus. Indeed, the encephalitis that was described by Leichtenstern, in 1890, is generally referred to in the literature as "influenzal encephalitis," and it may be recalled that Pfühl, and later Nauwerck in fresher material, asserted that they had demonstrated the presence of the *Bacillus influenzæ* of Pfeiffer in the diseased cerebral tissues in "influenzal encephalitis." Though the latter demonstration is at present less convincing than formerly, owing to the reopening of the question of the etiology of influenza itself and to the doubt cast upon Pfeiffer's bacillus as its cause,[1] still the occurrence of encephalitis in epidemic (and in pandemic) distribution in such close connection with epidemic (and pandemic) influenza as that observed in 1890–1892, and as that at the present time, strongly suggests at least one of the three following possibilities: (1) that the influenzal virus is the cause of the epidemic encephalitis, (2) that influenzal infection predisposes to infection with the virus of encephalitis, or (3) that the cosmic influences that determine the occurrence of epidemic influenza also determine the occurrence of epidemic encephalitis, even if each disease is due to a separate and distinct virus. It may be argued also that influenzal encephalitis tends to assume a special clinical form (acute cerebritis), but certainly other forms also occur in the epidemic; indeed, in the present outbreak, the polioencephalitis-superior type has predominated. Granting the weight of the arguments adduced, it must be admitted, however, (1) that the incidence of epidemic encephalitis is very low compared with that of influenza, (2) that the available data do not permit of the assertion of any direct parallelism of incidence and distribution in the two diseases, and (3) that some of the patients who have suffered from encephalitis deny any preceding attack of influenza, though doubtless all have been exposed to that disease. Thus until we are sure of the precise nature of the virus of influenza on the one hand and of the virus of epidemic encephalitis on the other, the questions regarding the exact etiological relationships of the two diseases must be left open.

It is but little wonder, too, that some have thought that the virus of epidemic encephalitis may be identical with, or a slight modification of, the virus (*Flexneria noguchii*) that is the cause of the Heine-Medin disease (poliomyelitis anterior acuta). For, in the first place, since Wickman's studies, cerebral, pontile, cerebellar, bulbar and meningeal forms of the Heine-Medin disease are certainly known to occur. And, in the second place, the pathological-histological lesions in the central nervous system and the findings in the cerebrospinal fluid in epidemic encephalitis and in poliomyelitis show a close resemblance. But against the view of identity there are many facts that may be urged. The onset of the paralyses is very different in the two diseases, since in poliomyelitis the paralyses are all there from the beginning, whereas in epidemic encephalitis the paralyses begin insidiously, often as ptosis or loss of power of accommodation, and gradually extend through a period of several days or longer to other muscles. Bilateral symmetry of the paralyses is more common in encephalitis, unilateral or asymmetrical paralyses in the Heine-Medin disease. The fever is usually slight and often more marked after the paralyses have developed in encephalitis; in the Heine-Medin disease the fever is, as a rule, highest before paralysis occurs. There is a much higher leukocytosis in the blood and also a higher cell count in the cerebrospinal fluid in the Heine-Medin disease than in encephalitis. The age-incidence is also different, since encephalitis is common at all ages of life, whereas the Heine-Medin disease shows a special predilection for childhood and adolescence. Again, a careful study of the distribution of the cases of encephalitis and of poliomyelitis in England has shown that the two series of cases are distributed independently of one another. And, finally, if there were identity of etiology one would expect more typical encephalitis cases in epidemics of poliomyelitis and more typical poliomyelitis cases in epidemics of encephalitis than we actually see. Some investigators, though fully granting that there may not be identity of etiology, suggest that the virus of encephalitis may be one special strain and the virus of poliomyelitis another special strain of a single microörganism, just as we now recognize Type I, Type II, and Type III, etc., of the *Pneumococcus,* or recognize that *Bacillus paratyphosus A* and *Bacillus paratyphosus B* are two strains producing similar but not identical diseases. In favor of this view may be adduced the findings of von Wiesener, of Vienna, who asserts that he has isolated an organism (globoid diplococcal form) from encephalitis and reproduced the disease in monkeys, and that this virus resembles closely that isolated by Flexner and Noguchi in poliomyelitis. A similar virus seems to have been grown by Wilson from Bradford's "acute infectious polyneuritis" cases, and with this virus it was also possible to reproduce in monkeys what they believed to be the disease they had under study. Cleland and Campbell assert that they have successfully conveyed the virus of the disease to the sheep, the calf and the horse. It is, of course, possible that just as we have a group of diseases (the leishmaniases) due to different strains of species of *Leishmania,* so we may have a group of diseases (the flexneriases) due to different strains or species of an organism (*Flexneria*), of which the virus of the Heine-Medin disease is one. But until more extensive virus studies and experiments have been made it would be unwise to look upon such a view as anything more than an interesting speculation.

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[1] Cf. Bloomfield, A., and Harrop, G., et al.

That epidemic encephalitis may, finally, be due to some causative agent neither identical with nor even a modification of any other virus is quite possible, and is, indeed, the view held by a number of those who have studied the disease. If this view turn out to be the correct one it will be interesting, later on, to find out whether the virus isolated by von Wiesener, that isolated by Wilson, or some entirely different filtrable (or unfiltrable) virus is the etiological agent.

TREATMENT OF EPIDEMIC ENCEPHALITIS.

The patients under our observation have evidently been of the milder type, as none of them died. Convalescence has been tedious in several of them, and in how far there will be residual phenomena in some, it is as yet too early to be sure.

At the onset of the disease, rest in bed, protection from external stimuli of all kinds, laxatives, bland diet and relief of headache and of pains would seem to be desirable.

In our experience, lumbar puncture, done for diagnostic reasons, relieved the symptoms so markedly in several instances that it was repeated at intervals as a therapeutic measure.

During convalescence, prolonged rest, careful nursing, a nutritious diet and mild hydrotherapy, electrotherapy and massage have been the only measures made use of. Complete recovery without residuals seems to be common.

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PLEURAL EFFUSION WITH INVERSION OF THE DIAPHRAGM PRODUCING AN ABDOMINAL TUMOR; TOGETHER WITH REMARKS ON ACUTE PULMONARY EDEMA FOLLOWING TAPPING.[1]

BY DAVID RIESMAN, M.D.,

PHILADELPHIA.

A NUMBER of years ago I made an autopsy at the Philadelphia General Hospital on a case of left-sided pleural effusion. On opening the abdomen I found in the left upper quadrant a large, smooth, tense tumor which upon further exploration proved to be a bag made by the inverted diaphragm and filled with pleural fluid. The incident made a deep impression upon me, but was not duplicated in my further experience as pathologist. I have, however, seen this tumor twice since then, clinically. Of the first case I cannot find any record; the second I saw a few months ago; it is as follows: The patient is a widow, aged seventy-seven years, who came under my observation in April, 1918. The salient points in her case are as follows: For years she has suffered with diabetes mellitus and chronic nephritis with moderate hypertension. She has diabetic cataract and retinal hemorrhages. When I first saw her, the heart was slightly enlarged; there was a soft systolic mitral murmur; the legs were somewhat edematous. The urine contained sugar up to 3.2 per cent.; and albumin from traces to 2.8 grams per liter. Ketones were inconstantly present. The quantity of urine

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[1] Read before the Section on Medicine of the College of Physicians of Philadelphia, October 27, 1919.